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Pulsatile cerebrospinal model with cardio-vascular coupling

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Abstract: In this paper we describe a model of the human intracranial space which is extended with the vascular system. We focus on a detailed modelling of the coupling between cerebral vessels and intracranial pressure (ICP). On the one hand, the model is based on a linearisation of the Navier-Stokes equation for the blood vessels and a subsequent geometric discretisation, leading to a distributed parameter approach of coupled ordinary differential equations. On the other hand, a concentrated parameter modelling approach is developed for the intracranial space which is subsequently extended by a production and resorption circuit for the craniospinal fluid. The intention of the model is to describe the transmission of pulsatile blood pressure waves from the cerebral vessels to the ICP, as it can be observed from clinical continuous intracranial pressure measurements. A good agreement of pulsatile ICP is shown in an in-silico study, compared to clinical continuous ICP measurements. An analysis of vascular impedance under variable intracranial compliance and for a non-symmetric Circle-of-Willis completes this study.

Keywords: Hydrocephalus, biomedical modelling, cranio-spinal, compliance, intracranial pressure, distributed parameter

1. INTRODUCTION

Hydrocephalus denotes a congenital or an acquired widening of the cerebral fluid space and is often accompanied with an increased intracranial pressure (ICP). About 10.000 Hydrocephalus diagnoses are made every year in Germany, of which about 100 patients will die every year (DESTATIS (2011)). The widening of the cerebral fluid space is due to a pathological accumulation of cerebrospinal fluid (CSF) and results from a dysfunction of CSF production and resorption. This CSF imbalance typically leads to an increased ICP, which can assume values as high as 110 mmHg (Aschoff et al. (1999)). A pathologically increased ICP will lead to, for example, a degradation in brain capacity, an unsteady gait or incontinence, if not treated properly. State-of-the-art therapy is the application of a shunt. Consisting of a catheter and a pressure relief valve, a shunt can be used to drain CSF and to release it to a different body compartment (preferably to the peritoneal cavity) (Elixmann et al. (2014)). A special form of Hydrocephalus treated with a shunt is the "Normal Pressure Hydrocephalus" (NPH). In a NPH patient, the ICP is not continuously increased, yet the patient suffers from the previously described symptoms. Therefore, the mean ICP cannot be used for the diagnosis of NPH. Current studies, however, show that the intracranial compliance is decreased in almost all Hydrocephalus patients, including NPH patients (Manwaring et al. (2004)). The decreased intracranial compliance is typically accompanied by a morphological change of the so-called P-waves. These P-waves are induced by the volume changes of the blood that is pumped into the intracranial compartment and are therefore synchronous to the beating of the human heart. Depending on the pathological state of the disease, the envelope of a P-wave can change and may hence be used for the clinical diagnosis of Hydrocephalus. The detailed transmission or coupling mechanisms that lead to the clinically observed ICP P-waves are still unknown. A detailed model that can describe the characteristic P-waves by, for example, their corresponding transfer functions is highly valuable in terms of giving additional insight to sensitive parameters and subsequent diagnostic implications. Furthermore, the model could be derived to test new control algorithms for an intelligent shunt implant that might use these morphological P-wave information in order to compute a reference value, which might then be used to set up an optimal intracranial compliance. A number of different modelling approaches are available from the literature, which in most cases aim at the explanation of Hydrocephalus occurance or a specific pathological state. In most cases, modelling attempts are based on the finite element method (FEM) describing either structural mechanics or computational fluid dynamics (CFD) and focus on the mechanical properties of the brain or the CSF bulk flow, respectively. The first FEM models were 2-dimensional, enabling the description of mechanical stress only qualitatively (Nagashima et al. (1987)). Newer three-dimensional models include the spinal channel and are able to compute displacements in the cerebral aqueduct in addition to mechanical stress/distension distributions (Linninger et al. (2009)). Besides complex geometrical distributed parameter modelling approaches (FEM models), models based on concentrated parameters can be used that may lead to analytic time-domain solutions. Recently, the effects of pulsatile dynamics have been incorporated into a Hydrocephalus model (Wilkie (2010)), with the goal of determining their role in the pathogenesis of Hydrocephalus. Another recent model was used to describe the pulsation of ICP under infusion tests (Sobey et al. (2012)). The model is of spherical-symmetrical structure and includes a connection between ventricles and subarachnoid space. The ICP pulsation is thereby

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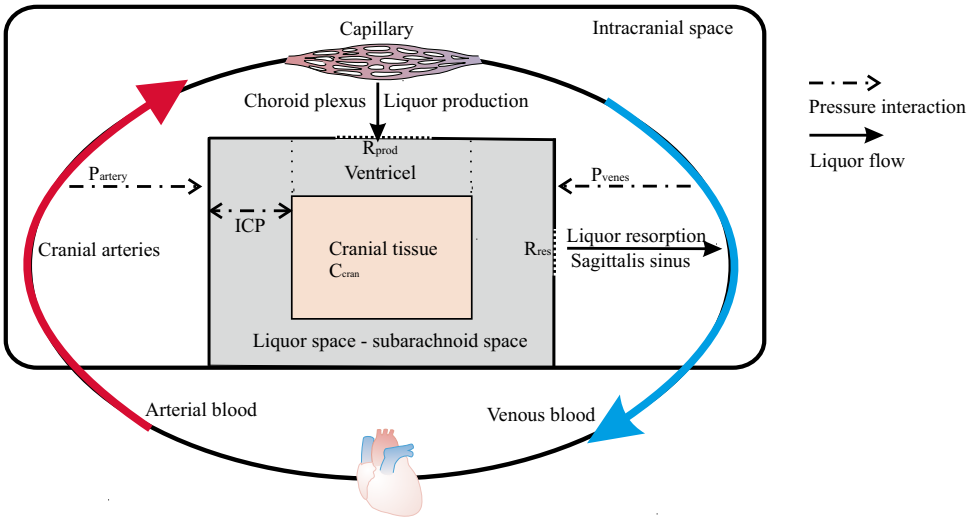

Fig. 1. Overview of the model showing the intracranial space and the heart.

realised by a spatially independent blood compartment which is elastically coupled to the CSF compartment.

In contrast to previous approaches, we present a model which consists of mixed distributed and concentrated parameter descriptions. The main intent of the model is to analyse the coupling between cardio-vascular pulsatile pressure and the intracranial space and thus to focus on a detailed modelling of the vascular system, connecting arterial and venous vessels to the intracranial space. The coupling between intracranial vessels and intracranial space is modelled with minimal complexity in this approach, with the goal of a reconstruction of the morphological shape of the intracranial P-waves from arterial pressure trajectories. The presented model is new in terms of a detailed distributed parameter arterial model, that is extended to the intracranial space to include the circle of Willis and connected to intracranial dynamics for the first time.

2. MODEL STRUCTURE

The basis of the vascular model is the work of Westerhof et al. (1969), which was later on extended by Avolio (1980) to a detailed model of the human arterial tree. In contrast to Avolio (1980), our model only includes the arterial system that is directly connected to the intracranial space. Furthermore, it is extended by a detailed model of the intracranial space that includes arterial bridging, like the circle of Willis (Cieslicki and Ciesla (2005)). The vascular model is then coupled to the intracranial space, described by a low order model of concentrated parameters and a CSF production/resorption circuit. In Fig. 1 an overview of the model is given. The blood is pumped from the heart through the ascending aorta and cranial arteries to flow back through capillaries and venes. Here, arterial and venous pressures in the intracranial space are coupled to the fluid space. The CSF circuit is implemented from a production in the intracranial capillaries through ventricles to the resorption in the sagittal sinus.

2.1 Vascular modelling

Blood flow in distensible vessels can be described by the Navier-Stokes equation (NSE). Denoting $\mathbf{p}$ as the pressure and $\mathbf{v}$ as the velocity of a fluid particle, the NSE for a Newtonian fluid can be given by

$$\begin{aligned}\rho\dot{\mathbf{v}} &= \rho\left(\frac{\partial v}{\partial t} + (\mathbf{v}\times\nabla)\,\mathbf{v}\right)\\ &= -\nabla\mathbf{p} + \eta\triangle\mathbf{v} + (\lambda+\eta)\,\nabla(\nabla\mathbf{v}) + \mathbf{f},\end{aligned} \tag{1}$$

where η describes the dynamic viscosity, λ is the Lamé-constant, ρ is the density of blood and $\mathbf{f}$ is an exogenous force. Furtheremore, viscosity and density of blood are assumed to be constant and thus, for an incompressible fluid, the continuity equation

$$\frac{\partial\rho}{\partial t} + \rho\nabla\mathbf{v} = 0, \tag{2}$$

simplifies to $\nabla\mathbf{v} = 0$. Thus eq. (1) simplifies to

$$\dot{\mathbf{v}} + (\mathbf{v}\times\nabla)\,\mathbf{v} = -\frac{1}{\rho}\nabla\mathbf{p} + \frac{\eta}{\rho}\triangle\mathbf{v}. \tag{3}$$

To obtain a simplified local representation, like the electrical analogue shown in Fig. 2, eq. (3) has to be linearised. Here it was shown by, for example, van de Vosse and Stergiopulos (2011) that the velocity profile of the pulse wave proceeds mainly in axial direction such that radial velocities can be neglected. With further assumptions on homogeneity, elasticity, isotropy and Hooke's law for the elastic blood vessel wall, the following partial differential equations are obtained

$$\begin{aligned}-\frac{\partial p}{\partial x_v} &= L\frac{\partial q}{\partial t} + Rq\\ -\frac{\partial q}{\partial x_v} &= C\frac{\partial p}{\partial t},\end{aligned} \tag{4}$$

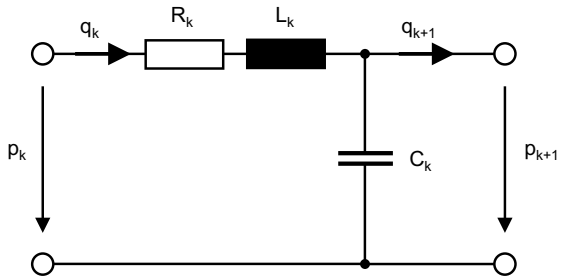

Fig. 2. Electrical analogue circuit for a single linearised compartment of the NSE of length Δl.